areas. Horowitz and Adams[7] also using patients with epilepsy, noted a high frequency of formed visual hallucinations following stimulation of the posterior dorsal hippocampus, the amygdala, the anterior hippocampus, the hippocampal gyrus, and in one case from the globus pallidus. In this study, speech was altered showing slurring, neologisms, slowing, blocking and semantic distortion, and the subjects experienced the phenomena as involuntary intrusions into their awareness. The authors implicated disruption of hippocampal functions in this process, disorganizing regulatory function over registration of sensation, the holding of a percept as an image, and the matching of it against memory images of prior perceptions.

Further evidence for the role of such structures in hallucinations comes from the observations of patients with complex partial seizures in whom psychosis identical to schizophrenia may develop. In particular, the special auditory hallucinations referred to above, pathognomonic of schizophrenia, are seen with dominant temporal lobe lesions[14]. In these situations however insight is lost and so some additional factor, not encountered in acute stimulation studies, is involved.

An important point here is to emphasize that allocortical, as opposed to neocortical, structures have been primarily linked to complex hallucinatory experiences, and their subcortical projections should be considered. For example, in the epilepsy model of psychosis, development of the latter has been well-described after temporal lobectomy[10], implying that the temporal lobe *per se* may not be the site of the most important changes.

Evidence of the role of subcortical structures in the pathogenesis of hallucinations comes from delirium, in which alteration of consciousness primarily occurs. The disintegration of cerebral mechanisms is usually associated with alterations in the reticular activating system[12]. In this situation, loss of insight for hallucinations is also noted. Within the reticular activating system lie important nuclear structures such as the locus coeruleus and the raphe nuclei, whose cell bodies form discrete pathways which proceed rostrally within the nervous system. Orthodox and paradoxical sleep are somehow associated with these systems, and it is during paradoxical sleep that dreaming, with all the hallucinatory experiences that it involves, occurs. The association between neurochemical events lucinogenic drugs, and there is extensive evidence that, for example, LSD acts primarily on serotonin-containing neurones[1]. In addition, breakdown products of several of the monoamines are themselves hallucinogens, and it has been suggested, for example, that dimethyltryptamine is involved in the aetiology of schizophrenia[17]. The role of dopamine cannot be ignored since it is clear that drugs that block dopamine receptors are antipsychotic, and in schizophrenia the action of these drugs is most associated with reduction of positive symptoms, which of course includes hallucinations and delusions[4]. In addition dopamine agonism experimentally activates REM sleep, an effect blocked by dopamine antagonist drugs. The fact that important dopaminergic pathways arise from the ventral tegmental area and pass rostrally to midbrain and orbital frontal cortex, with the known links of the latter to medial temporal structures, may be of importance in understanding the subcortical mechanisms that are involved in hallucinatory processes and are affected by temporal lobe stimulation. As seen from the summary in Table II, loss of insight, the hallmark perhaps of what Arnold called ideal insanity, probably requires subcortical involvement as opposed to just allocortical disruption. Whether chronic amygdaloid/hippocampal lesions lead to such change is unclear, but further exploration of hallucinatory phenomena, especially in patients with neurological disease such as epilepsy, is likely to be rewarding for our understanding of psychopathology, especially if attention is paid to the quality and

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